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BUMED NEWS LETTER

a digest of timely information

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TABLE OF CONTENTS

Circular Letters:

IMPORTANT NOTICE

The security classification of the News Letter has been changed, beginning with this issue, from "Restricted" to "Open." The notices concerning matters of a purely service nature and all circular letters are for official use only and should not be made known to unauthorized persons. All "Restricted" issues of the News Letter previous to this issue remain "Restricted" until notice to the contrary.

When is TB Inactive?: Ask any number of lay persons or physicians, "When is tuberculosis inactive?" and many of them will reply, "When it has calcified." This, however, is neither a complete nor an entirely accurate statement of the case.

It is certainly true that calcification often accompanies healing of a particular form of tissue destruction caused by tuberculosis, namely, caseation, but it is not an invariable accompaniment, nor is it a reliable index of healing. The reason it has been so widely regarded as a principal criterion of inactivity is that calcified deposits in the lungs are so frequently encountered as the sole residues of past tuberculous processes, particularly those of childhood.

Tuberculous infection in childhood, if it does not progress immediately to an overwhelming and rapidly fatal generalized form, is usually well withstood. In this age period calcification is also commonly a prominent accompaniment of healing, and in later life the only remaining evidence of disturbance may be one or more calcified nodules at the site of the former primary infection.

Such calcified nodules are generally regarded as of no immediate clinical significance when encountered later in life on routine roentgenographic examination. Pathological studies have shown, however, that the calcification may be incomplete and that there may remain a core of potentially active infection. Clinical experience has also shown that reactivation of such residual foci may occur and disseminate infection to other parts of the body.

For these reasons workers in tuberculosis long ago ceased to place much emphasis, if indeed they ever did, on calcification as an index of inactivity of tuberculous disease. Many tuberculous processes may become inactive, or even entirely healed, without the deposition of any calcium in the tissues.

What, then, can be used as a criterion of inactivity? It is well known that absence of symptoms is not a reliable guide. Patients usually are not permitted to return to work or resume other normal activities until many months after their symptoms have entirely cleared. Many may never have had symptoms and yet are advised to give up their usual lives and go to bed for extended periods. Indeed, the aim of all the campaigns for early diagnosis by means of routine x-ray examinations has been to discover active disease before there are any symptoms.

Except in instances of advanced disease, physicians cannot tell by stethoscopic examination whether a tuberculous focus is, or is not, present, but even if they were able to determine the presence of a focus, they could not state whether or not it was active. The presence of bacilli on examination of sputum or other material will, of course, establish that the disease is active, but their absence by no means establishes the contrary. Other laboratory tests are similarly inconclusive.

Skin testing with tuberculin might be looked to as a possible help. Practically, however, it has not contributed significantly to the solution of this

problem. There is some evidence that the tuberculin reaction may persist even after bacilli have died out and, in any case, complete eradication of infection is so rarely attained that even were there a reliable index of such sterilization of the body it would be of little or no practical value.

From the standpoint of roentgenographic interpretation it is always difficult, and often impossible, to determine from a single examination whether tuberculosis of the lungs is active or inactive. Repeated examinations over a period of many months, on the other hand, usually answer the question, since an active disease process in the lung generally causes some change in the pattern of abnormal shadows over such a period of time. Once the active foci have been isolated and encompassed by the body's defensive tissue reactions, the shadows no longer change and, roentgenographically, the abnormal pattern, though it does not disappear, becomes stabilized.

The answer to the question, "When is tuberculosis inactive?", therefore, cannot be made categorically. In practice it is a matter of somewhat arbitrary classification and definition. The classification offered in Diagnostic Standards of the National Tuberculosis Association takes into account all of the above mentioned factors and has been accepted by all clinicians specializing in tuberculosis.

The terms, "quiescent," "apparently arrested," "arrested," or "apparently cured," used as diagnostic classifications in connection with discharging patients from sanatoriums or from clinics, have definite meanings with respect to the length of time since bacilli were demonstrable. They also have definite implications with respect to how long patients have been free of all symptoms, how long repeated x-ray examinations have shown a favorable condition and how much physical activity the patient has been capable of without any reversal of favorable trends.

The terms used to indicate the clinical status of a patient, as defined by the National Tuberculosis Association, are as follows:

"Apparently Cured - Constitutional symptoms absent. Sputum, if any, must be found negative for tubercle bacilli, not only by concentration and microscopic examination, but also by culture or animal inoculation. In case there is no sputum, the fasting gastric contents should be obtained and similarly examined. Lesions stationary and apparently healed according to x-ray examination. These conditions shall have existed for a period of two years under ordinary conditions of life. A considerable but undetermined percentage of apparently cured patients particularly those who have fulfilled the above requirements not only for two, but for six years, may in regard to their survival expectancy (as to tuberculosis) reach normal standards.

"Arrested - Constitutional symptoms absent. Sputum, if any, must be concentrated and found microscopically negative for tubercle bacilli. Lesions stationary and apparently healed according to x-ray examination; no evidence of pulmonary cavity. These conditions shall have existed for a period of six months,

during the last two of which the patient has been taking one hour's walking exercise twice daily, or its equivalent.

"Apparently Arrested - Constitutional symptoms absent. Sputum, if any, must be concentrated and found microscopically negative for tubercle bacilli. Lesions stationary and apparently healed according to x-ray examination; no evidence of pulmonary cavity. These conditions shall have existed for a period of three months, during the last two of which the patient has been taking one hour's walking exercise daily, or its equivalent.

"Quiescent - No constitutional symptoms. Sputum, if any, may contain tubercle bacilli. Lesions stationary or retrogressive according to x-ray examination; cavity may be present. These conditions to have existed for at least two months during which time the patient has been ambulant.

"(Frankly) Active - Improved, Unimproved - Symptoms unchanged, worse or less severe, but not completely abated. Lesions not completely healed or progressive according to x-ray examination. Sputum almost always contains tubercle bacilli."

Significantly, Diagnostic Standards, published by the National Tuberculosis Association, includes no classification of "cured." The omission is deliberate and recognizes the fact that, though patients may have been "apparently cured" for many years, the infection is usually not eradicated, but merely controlled. Clinical relapse is always possible and for this reason patients should remain under medical observation with periodic examinations which will generally reveal any sign of reactivation of the disease before it has had opportunity to progress seriously, provided that the examinations are made at stated intervals appropriate for the particular case. (Bull. Nat'l TB Assoc., Oct. '47 - C. Muschenheim)

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Recovery of Ornithosis Virus from Pigeons in Baltimore, Maryland: The infection of pigeons (Columba livia) with a virus of the psittacosis-lymphogranuloma venereum group was first reported in South Africa by Coles and in the United States by Pinkerton and Swank in 1940. Later Meyer related the infection to human disease by isolating virus from a flock of racing pigeons and the same virus from the lung of a human who had been exposed to the birds and who developed a fatal pneumonitis. The name "ornithosis" has been proposed for the disease contracted from birds other than psittacine birds in order to distinguish it from psittacosis.

Although nearly all reported cases of ornithosis in which the source of the virus could be traced had been contracted from privately owned pigeons, there remained the question of possible danger to humans from exposure to pigeons present in or about public parks, squares, or buildings. In order to secure data on which to estimate this potential danger, an investigation of the infection among such pigeons in Baltimore, Maryland, was undertaken in 1945.

Only two cases of ornithosis were reported to the Baltimore City Health Department in the years 1944 and 1945, but it is possible that some human infections with this virus may not have been recognized. The first case was that of a 38-year-old white female who became ill on 6 July 1944 with evidence of a severe pneumonitis. Her serum fixed complement in the presence of psittacosis antigen. The patient's son raised pigeons in the back yard of their home. Ornithosis virus was isolated from two of these pigeons at the National Institute of Health.

The second reported case was in a 43-year-old colored male who had onset of disease on 1 September 1945, and exhibited the signs and symptoms of a moderately severe pneumonitis. His serum taken during the third week of illness fixed complement in a dilution of 1:256 in the presence of psittacosis antigen. The patient's son raised pigeons in the back yard of his home but was not sick himself, and his serum did not contain demonstrable antibodies against psittacosis virus. Pigeons from this loft could not be secured for examination.

The fixation of complement in the presence of psittacosis antigen by serum from an infected bird has been a simple and useful method of estimating the number of infected pigeons in a given flock. However, virus has been isolated from birds whose serum was negative, possibly because the serum had been tested in the early stages of infection. Attempts at isolation of virus have frequently been unsuccessful in birds whose serum reacted positively, probably indicative of a past infection.

Although details of the mode of excretion of the virus from pigeons are not completely known, isolation of infective virus from birds would appear to yield more definite information concerning their infectivity than the reaction of serum. Actual isolation of the virus, therefore, was attempted from each of 100 adult pigeons trapped at various locations in the city of Baltimore. A virus belonging to the psittacosis group was recovered from 15 of the pigeons examined. Infected birds were found in four of eight separate locations in the city. This would suggest that the infection is ubiquitous throughout the city.

The spleens of all pigeons subsequently shown to be carriers of the virus were enlarged, but no other obvious abnormality was observed in any of the birds examined. A number of birds from which no virus was recovered had enlarged spleens.

Four virus strains were inoculated into the yolk sac of developing chick embryos. All grew readily, and three of these, after being established by several yolk-sac passages, killed mice through intraperitoneal and intracerebral injection with 50-percent end points. It was apparent that the virus was from 10^5 to 10^7 times as lethal when injected into the brain of a mouse as when injected into the peritoneal cavity. One strain of virus was recovered from the spleen of a mouse inoculated intraperitoneally 50 days previously, indicating a carrier state for at least this time period.

Antigen prepared from these three strains by ether extraction of heavily infected yolk sacs, after the method used for rickettsial antigens, fixed complement

in the presence of human psittacosis serum, pigeon ornithosis serum, and pooled human lymphogranuloma venereum serum, but not in the presence of normal human serum.

In spite of the demonstrated prevalence of infection among these pigeons, the paucity of recognized human ornithosis suggests that such pigeons do not constitute a serious public health hazard to the population of Baltimore under conditions now prevailing in that city. (Pub. Health Reps., 10 Oct. '47 - D. J. Davis and C. L. Ewing)

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Use of Penicillin in the Treatment of Carriers of Beta-Hemolytic Streptococci Among Patients with Rheumatic Fever: The danger of hemolytic streptococcus respiratory infection to persons who are susceptible to rheumatic fever is well recognized. Since, with present knowledge, there are inherent difficulties in evaluating the innocuousness of a given strain, every carrier of beta-hemolytic streptococci must be considered a potential menace to a population of rheumatic-fever patients. Prolonged isolation of such carriers is inadvisable from the psychologic standpoint and is usually impracticable. Hence, means of abolishing the carrier state are desirable.

During the administration of sulfonamides to carriers the throat cultures become negative for hemolytic streptococci in some cases, whereas in others the cultures, although remaining positive, usually yield only small numbers of the organism. In most cases, however, when therapy has been discontinued, nose or throat cultures again become strongly positive.

Many investigators have found penicillin to be a more effective agent than sulfonamides for the treatment of hemolytic streptococcus respiratory infections. From the reports it also seems that during penicillin therapy hemolytic streptococci disappear from the nose and throat in nearly all cases. After the discontinuation of treatment the organisms have reappeared in a varying proportion of the reported cases, depending apparently on the dose of penicillin and the number of days of treatment.

With a view to preventing the spread of hemolytic streptococci from one patient to another, since November, 1945, penicillin has been administered to all patients at the House of the Good Samaritan (Boston) found to have positive throat cultures.

All patients whose routine throat cultures yielded hemolytic streptococci were followed by means of cultures taken at least three times a week until the presence of the carrier state was established - that is, until the number of positive cultures was shown to be well in excess of the number of negative cultures, with no tendency to diminish.

Any patient suffering from an acute streptococcal infection was placed under treatment with penicillin as soon as the diagnosis was made. A control culture

was taken just before the first dose of penicillin. In such cases the treatment was identical with that given the healthy carriers.

The organism isolated in each case was grouped and typed by the Lancefield capillary precipitin technic, and its sensitivity to penicillin determined.

Penicillin in doses of 10,000 units every two hours, day and night, for ten days was given intramuscularly to a total of 1,200,000 units to all patients in these two classifications, that is, 18 chronic carriers of beta-hemolytic streptococci and 2 patients with acute streptococcal tonsillitis. Throat cultures were taken three times weekly during treatment, daily from one to two weeks after treatment and then with gradually decreasing frequency to a routine weekly schedule. Antistreptolysin O titers and erythrocyte sedimentation rates were followed at bi-monthly intervals.

In 17 cases the infecting organisms were eliminated permanently from the throat cultures. One Group A streptococcus and two that did not belong to Group A, C, or H failed to disappear. In 2 cases the antistreptolysin O titers did not rise after acute streptococcal infections that were promptly treated with penicillin.

When tonsillectomy was clinically indicated in the penicillin-treated cases, it was performed after the rheumatic fever had subsided and was preceded by several daily throat cultures, the last immediately before operation. These operations were done on five patients from 125 to 185 days after treatment. Cultures of the open tonsillar fossae were obtained during operation, and the tonsils were sectioned and cultured. They were both found to be free of hemolytic streptococci. The patients were given penicillin prophylactically, beginning immediately after operation and continuing until the operative wound was healed and there was no fever.

A program similar to that described above but using a slowly absorbable penicillin preparation was undertaken at a convalescent home, where repeated rounds of streptococcal infections had been troublesome. Two sets of throat cultures of patients and personnel were obtained within a few days of one another, and in each case in which beta-hemolytic streptococci were found in one or both of these cultures, two additional cultures were obtained at intervals of a few days. Treatment was instituted in the 6 cases in which these preliminary cultures were positive. Penicillin in beeswax and peanut oil was given to each of the selected patients intramuscularly in single daily doses of 150,000 units for ten days, to a total of 1,500,000 units. Throat cultures were taken at intervals of three or four days, beginning twenty-four hours after the first dose of penicillin, that is, just before the administration of the second dose. Serum penicillin concentrations were determined at specific intervals.

In 5 of the 6 cases the cultures remained free of hemolytic streptococci for the duration of the observation period (about one month).

The ability to eliminate beta-hemolytic streptococci from the throats of most patients in close contact with persons who have had rheumatic fever suggests a practical method of protecting the latter group from beta-hemolytic streptococcus infection.

It is evident that further observations are needed to ascertain whether prompt penicillin treatment for acute hemolytic streptococcus infections in subjects with rheumatic fever may decrease the likelihood of a recurrence of rheumatic fever. (New England J. Med., 16 Oct. '47 - J. R. Goerner et al.)

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Pitressin Test of Coronary Insufficiency: Pitressin has been shown by numerous investigators to be one of the most powerful vasoconstrictors, particularly of the coronary arteries. Because it has been shown that pitressin produces a marked diminution of the coronary artery inflow in anesthetized dogs, the use of this drug suggested itself as a test of latent coronary insufficiency, to differentiate true angina pectoris from other conditions associated with chest pain. The manufacturers have strongly urged that pitressin, in ampoules of 1.0 c.c. containing 20 pressor units each, be used only subcutaneously or intramuscularly, and not intravenously. Parenteral administration of the usual doses of pitressin is not infrequently accompanied by unpleasant nausea, abdominal cramps, and a desire to urinate and defecate. It is for this reason that the electrocardiographic effects in human beings from the use of pitressin have not been extensively studied.

Patients considered to have angina pectoris and also patients whose suspicious symptoms were thought to be nonanginal in type were selected for study; those with recent myocardial infarction or congestive heart failure were carefully excluded. Slowly increasing doses of pitressin were used, at first, intramuscularly, and finally, intravenously, in preliminary studies. For a subject of average weight (70 kilograms), generally satisfactory dosages were found to be 2 c.c. (40 pressor units) intramuscularly or 0.75 c.c. intravenously injected in about 60 seconds. Dosages were correspondingly adjusted for marked weight deviations. In later studies the intravenous route has been used almost entirely. Slow injection and, in cases of suspected severe coronary insufficiency, smaller preliminary parenteral dosage, are of paramount importance in preventing possible serious myocardial mishaps.

Following the injection of pitressin, the systolic and diastolic pressure usually rose 20 and 10 mm. Hg respectively, sometimes fell temporarily (that is, with bradycardia, or remained constant. Electrocardiographic changes, even in those patients with evidences of marked myocardial damage to begin with, have been limited, at most, to marked sinus bradycardia, with or without A-V nodal escape beats, premature contractions usually of ventricular origin, and S-T and T-wave shifts characteristic of coronary insufficiency.

Slight precordial pain or burning ache was produced in two patients with clinical angina pectoris. A few instances of severe gagging with occasional

slight vomiting were encountered in those who had just eaten and had received no preliminary sedation. Care must be taken not to employ coronary vasodilators for this purpose. Sodium pentobarbital or seconal in doses of 0.1 Gm. each were found to be satisfactory in the few instances in which they were required for proper repetition of the test. An empty stomach is a useful, but not necessary, prerequisite. All patients were able to control their desire to urinate and defecate. The abdominal cramps, when present, were slight. Pallor and coldness and, at times, burning of the face usually occurred as an immediate result of the intravenous administration of pitressin.

The time intervals for the appearance of significant electrocardiographic changes following intravenous pitressin were empirically determined to be immediately after the injection was completed, and 2, 6, 10, and 14 minutes later. The same intervals may be used following intramuscular injection. Changes usually occurred within from 6 to 10 minutes, and reverted to normal after from 10 to 14 minutes. Leads I, II, III, CF_2 and IVF were uniformly employed with the patient in the horizontal position, both for the control and pitressin readings.

Twelve patients with definite clinical angina pectoris on a coronary arteriosclerotic basis and 10 of suspicious but indefinite "cardiac" pain in association with hypertension, arteriosclerosis, syphilis, and anemia were tested both by the pitressin and the Master exercise tests, for purposes of comparison. Five normal controls had the pitressin test only.

Positive electrocardiographic indications of coronary insufficiency for both the pitressin and Master tests were taken to be: (1) Change from positive T_1, T_2, or T_4 to a flat, diphasic, or negative T wave. (2) The sum of deviations (almost always downward) of the S-T segments from the controls of 3 mm. or more in Leads I, II, III, and IVF. These criteria follow the more rigid requirements of the anoxemia test of Levy and associates rather than those of the "two-step" exercise test of Master and associates. Extensive experience with the Master test has forced the author and associates to adopt the more rigid criteria for it also. In a patient with a previous myocardial infarction, $S\text{-}T_4$ elevation was prominent. In two patients with clinical angina pectoris T waves in Leads II and CF_2 changed from positive to diphasic. Such changes were not seen in any of the patients whose chest pain was considered to be nonanginal or in the normal controls.

Of the 12 patients clinically diagnosed with fair certainty as having angina pectoris, 5 showed positive pitressin and Master exercise tests. Three others showed a positive pitressin test and a negative Master test. Two patients showed a positive Master test and a negative pitressin test; in one of these 2 patients the pitressin dosage (1 c.c. intramuscularly) was probably inadequate. The remaining 2 patients, both of whom had definite clinical angina pectoris, showed negative Master and pitressin tests. All of the patients in this group who gave negative pitressin tests showed unfavorable electrocardiographic changes, such as depression of the S-T segments, but the changes were insufficient to satisfy the preceding rigid criteria of coronary insufficiency.

All 10 patients whose indefinite symptoms were not thought to be due to true angina pectoris had negative Master and pitressin tests. Some of the patients in the nonanginal group who gave negative pitressin tests presented possible favorable electrocardiographic changes, such as higher voltage of the T waves, following both the Master and pitressin tests.

Repeated pitressin tests, provided that the same dosage was used and provided that the tests were done on different days, reproduced the same results almost exactly in most patients. Repetitions of the drug in animal experiments results in loss of the coronary vasoconstrictor effect. This should be kept in mind if repeated tests are contemplated.

The results appear to establish the use of pitressin as a valid test of coronary insufficiency. The pitressin test is apparently equivalent to the Master exercise test, and has the advantage that it may be used in patients unable to exercise. Even standardized exercise has extremely variable effects in the production of cardiac pain, as well as of electrocardiographic changes. This may be also true of the hypoxemia test, which has, moreover, occasionally resulted in some serious cerebral and other sequelae. In patients showing marked cardiac acceleration after exercise or hypoxia, the effect on the ventricular gradient may possibly cause positional S-T and T-wave changes simulating those of coronary insufficiency. In these patients, too, the pitressin test would seem to be of value.

The author and associates were forced by dearth of available cases of angina pectoris to study the effects of pitressin in some patients who already presented definite electrocardiographic evidences of myocardial damage. The interesting fact, therefore, is that the same criteria of positivity and negativity of the pitressin and Master tests can apparently be applied to these patients with abnormal tracings, as well as to patients whose electrocardiographic patterns are originally normal. This may also indicate that even in cases of marked coronary sclerosis, spasm of some arterial branches is still possible. Thus, in the group with clinical angina pectoris there were 5 patients in whom control or previous tracings presented T-wave negativity, S-T depression, and even left bundle branch block. Two of these, including the patient with bundle branch block, showed identical positive pitressin and exercise tests; 2 others had positive pitressin and negative Master tests; and one had the opposite findings. In the clinical group without real angina pectoris, three patients with originally abnormal electrocardiograms responded negatively to both pitressin and exercise, as did those presenting normal control electrocardiograms.

Previous students of tests for coronary insufficiency have emphasized the need for excluding digitalis-treated patients, since digitalis modifies the electrocardiogram in the same general way as the tests do. Two patients with clinical angina pectoris who had abnormal electrocardiograms had been in congestive failure, and were taking maintenance doses of digitalis leaf (0.1 Gm. per day) at the time the tests were made. In one, the pitressin and Master tests were both positive; in the other, the Master test was positive, the pitressin test (1 c.c. intramuscularly, inadequate dose?), negative. Further studies will be necessary

to show that control electrocardiograms modified by the effects in patients of digitalis do not actually interfere with tests of coronary insufficiency. On the other hand, latent coronary insufficiency not due to coronary disease, as in severe anemia (two cases cited), apparently is less liable to become manifest in the exercise or pitressin tests. The electrocardiographic changes in the 2 cases of anemia were suggestive but not diagnostic of coronary insufficiency by the criteria of the author.

Patients with systolic (up to 200 mm. Hg) or diastolic (up to 120 mm. Hg) hypertension, of which there were 6 in the angina group, and 4 in the nonangina group, showed a response that was similar to the response shown by nonhypertensive patients. The effects of pitressin were no more untoward in the hypertensive patients than in others, even though the blood pressure, especially the systolic pressure, usually rose somewhat higher than in patients with normal blood pressure. Positive pitressin tests were obtained in those rare cases in which the blood pressure fell (for example, from 160/100 to 80/40 temporarily in one case), as in the majority in which it rose. Apparently, the coronary constrictive effect of pitressin overcomes any increase in coronary flow which may be due to a rise in aortic blood pressure.

Although there is some disagreement concerning the degree to which pitressin affects the heart through the vagus nerve, direct myocardial effects (for example, modification of cardiac output) have practically been excluded for nontoxic doses in animals. In man, pitressin actually decreases the cardiac output in the first 10 minutes after its parenteral injection or during the approximate duration of the electrocardiographic changes which have been described. This is in direct contrast to epinephrine, which was formerly used as a test for relative coronary insufficiency on the basis that it increased the cardiac work to a greater extent than it increased the coronary blood flow. Exercise presumably has the same effects, and the "two-step" test the same basis, as the epinephrine test. Even eating apparently involves increased cardiac demands sufficient to produce electrocardiographic abnormalities in cases of coronary slcerosis. Instead of such an indirect effect, pitressin produces relative myocardial ischemia through direct coronary vasoconstriction, in which it excels other similar drugs.

Positive tests for coronary insufficiency confirm the clinical suspicion of coronary pain; negative tests do not exclude it.

The side-effects of pitressin render its general use as a test for latent coronary insufficiency inadvisable. In the hands of experienced investigators it may be useful in the evaluation and management of coronary sclerosis, particularly in younger individuals, and for experimental purposes. Pitressin must not be used without thorough training in its pharmacologic effects in ascending doses and in the estimation of the degree of coronary insufficiency by all other modern methods. Otherwise serious and even fatal myocardial ischemia and necrosis may result.

The results of the use of pitressin attest both coronary spasm and relative myocardial ischemia as theoretical bases for clinical anginal pain. (Am. Heart J., Oct. '47 - A. Ruskin)

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Preliminary Report on Experimental and Clinical Studies with Polythene Film: The effort to find an adequate substitute for the dura mater, when this membrane has to be sacrificed because of injury or disease, has occupied investigators for many years.

There is general agreement that the dural substitute should possess the following basic characteristics: (1) inertness in cerebral tissue, (2) nontoxicity, (3) nonresorbability, (4) high tensile strength, (5) elasticity, (6) ability to hold sutures, and (7) nonadherence to leptomeninges and cortex.

The materials which have attracted the most attention during the recent war period as dural substitutes are tantalum foil, fibrin film, and animal membranes. Although they are valuable adjuncts in certain situations, none of these substances completely fulfills all the criteria for artificial dura. All are notably deficient in ability to hold sutures and have many individual drawbacks with respect to the other properties that have been mentioned.

When polythene film became commercially available in quantity and appeared to possess the requisite physical properties for an artificial dura, experimental tests were instituted to determine its suitability. For the past year and a half it has been used at the Institute of Experimental Medicine for the surgical reconstruction of several organs. These experimental studies are still in progress, but it is not believed that it is too early to state that polythene has great promise as a material for temporary or permanent surgical implantation in living tissue.

Polythene, or polyethylene, is one of the more recently developed plastics. It is made by polymerizing, under high pressure, molecules of ethylene. The result is macromolecules consisting of long carbon chains in which each carbon bears two hydrogen atoms. Polythene is simply a glorified paraffin in which the chain molecules are very much longer than in ordinary household paraffin. Like paraffin, polythene is insoluble in, and is not wetted by, water or aqueous solutions. It is unaffected by strong acids and alkalies and by powerful reagents such as fluorine gas.

Unlike paraffin, polythene is completely insoluble in all known solvents at a temperature of 60° C. or less, nor does it melt in boiling water. Polythene is flexible, elastic, and much tougher than paraffin, and its softening point is at a temperature greater than that of boiling water (approximately 110° C.) It is light in weight (specific gravity 0.92), conducts heat poorly, is an excellent electric insulator, and has a low coefficient of friction. It is transparent to roentgen rays. Polythene has a pearly gray color, but in thin sheets it is transparent and colorless. Unlike cellophane, polythene holds a suture very well; that is, a suture

placed near the edge of thin film does not tear out. Other virtues of polythene are that it is cheap, that it is easily sterilized by chemicals or boiling in water and that it is easy to handle. It can be cut to a desired shape with knife or scissors, and by heating it to its molding temperature, molded articles of polythene can be made.

The authors have found no evidence of reaction (foreign body or inflammatory), when polythene is buried in body cavities or tissues. A method of inserting capillary-sized tubing made of polythene into veins for continuous intravenous administration has already been reported. In some cases it has been possible to keep a solution for intravenous use running through such a tube into the same vein for two weeks. The authors state that in the near future they shall describe methods of anastomosing the common bile duct, trachea, and pelvic colon respectively with molded polythene tubes. In these studies, also, they have observed no tissue reaction to polythene and no tendency for the plastic to deteriorate or for the tubes to become blocked with secretions or solid material.

Ten animals (8 dogs and 2 monkeys) were employed in the experimental study with polythene film and tubing. All operations were carried out under intratracheal ether anesthesia, sterile technic being used. In 7 animals large osteoplastic bone flaps were fashioned and a comparable area of dura was excised and replaced with polythene film; in addition, in two of these animals the underlying cortex was lacerated to produce specific neurologic defects. In the remaining three animals polythene tubes were inserted into the lateral ventricles to form an artificial fistula between these cavities and the subarachnoid space. The brains of eight animals have been re-examined at intervals of two, four, and six months; the others will be observed indefinitely for possible late deleterious effects. Wound healing has been normal in all instances, and there have been no deleterious neurologic effects attributable to the artificial dura. In the two instances in which the cortex was lacerated beneath the film, recovery of sensory-motor function proceeded at the anticipated rate. All ventriculostomy tubes have been re-examined and found to be patent and unchanged in appearance.

Examination of the polythene film after removal from the animals showed that no changes in its physical properties had occurred. The film was entirely nonadherent to the underlying leptomeninges and cerebral cortex, and was surrounded by a small dead space. Grossly and histologically there was a thin subdural neomembrane that had formed between the film and the arachnoid; this was actually a regeneration of the inner layer of the dura mater. The collagenous fibrils of this neomembrane were oriented, in regeneration, in a direction that paralleled the film in the dead space. The neomembrane was likewise nonadherent and was easily dissected off the underlying arachnoid in both the wounded and unwounded animals; there appeared to be no tendency for a downgrowth of fibrils to form a traction cicatrix. Histologic sections have shown no evidence of reaction in the underlying arachnoid, pia, and cortex.

A preliminary clinical trial of polythene film indicates that the material is a satisfactory substitute for the dura mater when repair or replacement of this

membrane is necessary. By utilizing the various properties of polythene, it is possible to facilitate many procedures in the everyday practice of neurosurgery. The film is primarily intended to replace the dura when excision is necessary in invasive tumors, such as meningioma, and to repair dural defects in cerebrospinal rhinorrhea and other spinal fluid fistulas. Subsidiary uses of polythene film which may prove to be of increasing importance are the following: (1) the prevention of meningocerebral adhesion in penetrating craniocerebral wounds, (2) to provide both protection and elasticity for the herniating lobe in subtemporal and suboccipital decompression, (3) in peripheral nerve anastomosis to protect nerve ends and to surround the gap with a tube in two-stage procedures, (4) the prevention of damage to the arterial intima in ligations for intracranial aneurysm and (5) cosmetic uses, such as the covering of the anterior burr hole in transfrontal craniotomy. (Proc. Staff Meet., Mayo Clin., 1 Oct. '47 - M. H. Brown et al.)

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Distribution of Parietal Cells in Gastric Disease: There is now general agreement that the source of the hydrochloric acid of the stomach is the parietal cells of the gastric mucosa. Although the determination of gastric acidity has become almost a routine procedure in the study of stomach disorders, particularly in cases of peptic ulcer and cancer, the state of the cells which secrete the acid has been left largely to conjecture.

An attempt was made to determine whether there are quantitative or qualitative differences in the parietal cells in conditions in which there is usually hyperacidity (peptic ulcer) as contrasted with conditions in which the acid is usually low or absent (gastric cancer). The problem is of more than academic interest, since Hurst and others have reported that the absence of acid found so frequently with gastric carcinoma usually, if not always, precedes the onset of the malignant process.

A series of 200 stomachs surgically resected for gastric carcinoma or for peptic ulcer of the stomach or the duodenum was examined. The great majority of specimens were from patients from 40 to 60 years of age. The ages of the patients with duodenal ulcer averaged 47 years; those of the patients with gastric ulcer, 51 years, and those of the patients with carcinoma, 55 years. Males predominated in a 5 to 1 ratio.

In all specimens the number of parietal cells diminished as the pylorus was approached and was somewhat less along the lesser curvature than in corresponding areas on the walls or greater curvature.

The only quantitative change of significance was that in many cases of carcinoma there was a diminution of the number of parietal cells in the body and the fundus of the stomach, whereas in cases of peptic ulcer, especially cases of duodenal ulcer, such a diminution was less frequent.

A reduction of the number of parietal cells was not a constant finding in cases of cancer of the stomach; in many cases of cancer and complete anacidity there was an abundance of parietal cells. No stomach showed complete absence of such cells.

As seen in routine stains, there were no qualitative changes in individual parietal cells which could be correlated with ulcer or with cancer.

According to Best and Taylor, 60 per cent of cases of gastric carcinoma show complete absence of acid. It is somewhat surprising, then, that the acid-forming cells do not show fairly constant numerical or morphologic changes in cancer. Since the only change observed was a tendency for the parietal cells to be fewer and since abundant parietal cells were at times found with cancer and complete anacidity, one must look further for explanations of the problem.

Numerous theories have been proposed to explain diminished gastric secretion in the presence of cancer. One of the more recent is that of Brunschwig, who suggested that the cancer forms a "secretory depressant." Other explanations which have been offered include neutralization of gastric juice or suppression of secretion as a result of the action of the duodenal contents or mucus, interference with gastric nerves, chloride starvation and chronic gastritis. Polland and Bloomfield reviewed such explanations and concluded that "one can hardly evade the supposition that. . .gastritis may be the direct cause of the defective secretion." This is a view held by numerous observers and one which is usually reached by a process of exclusion rather than by positive findings. It is true that it has been shown that the incidence of chronic gastritis is higher in the fundus and the body of the stomach in cancer than it is in ulcer, and it is easy to assume that the diminution of the number of parietal cells which was found may be related to chronic gastritis. However, there is still left the difficulty of explaining anacidity in the presence of numerous parietal cells and in the absence of gastritis. Moreover, gastritis is a common condition and may be associated with hyperacidity as well as with diminished acid secretion.

It would seem, then, that there are probably several methods of bringing about the diminished secretion of acid which is seen in the presence of cancer of the stomach and that further investigation is warranted to determine more clearly the causes of this phenomenon and its relationship to the development of gastric cancer. (Arch. Path., Sept. '47 - W. A. Meissner)

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The Value of the Methylene Blue Test in the Detection of Bilirubin: When Fellinger and Menkes described a modification of Franke's methylene blue tes for bile in the urine, they suggested that certain quantitative potentialities of the test existed. They observed that determinations made on 24-hour collections of urine paralleled blood bilirubin levels to a satisfactory degree. Determinations on individual samples, however, were not reliable, since dilution factors, as represented by varying specific gravities of the urine, affected the depth of color that took place after the addition of methylene blue. The depth of color in their technic represented the basis of quantitative interpretation.

Gellis and Stokes, after considerable investigative studies, reported a further modification of the original test. They suggested that it might be used as an aid in the early recognition of bilirubinuria in infectious hepatitis. The authors did not claim to describe an unusually sensitive or accurate test for the presence of bilirubin but rather to suggest that the test could be used for demonstrating bilirubinuria in working with large numbers of soldiers in the field.

The present investigation was undertaken to determine the accuracy and reliability of the test as judged by civilian medical standards, in an effort to determine whether it has any place in routine laboratory procedures. The technic used was that described by Stokes and Gellis. This consisted of a drop-by-drop addition, to 5 c.c. of urine, of an aqueous methylene blue solution (0.2 per cent by actual dye content) from a 1-c.c. pipette delivering 20 drops per cubic centimeter. If 5 or more drops were necessary to change the green color of the mixture of urine and methylene blue to a blue color, the test was considered positive for bilirubinuria.

It was found that the test is nonspecific and may give falsely positive results when the urine contains yellow pigments, such as penicillin and riboflavin. The test is not significantly affected by changes in the urinary reaction, but is affected by changes in the specific gravity. It is less sensitive than the Naumann concentration test, the Harrison, the spot diazo, and the Watson modification of the Harrison and the foam tests. The Naumann concentration test, however, may give positive results in normal urines, and the foam test is highly nonspecific. The present study suggests that the methylene blue test is positive when the concentration of bilirubin in the urine is 2 or 3 mg. per 100 c.c. or more. Although the test is neither highly accurate nor specific, it provides a valuable means for estimating the daily excretion of bilirubin in the urine of jaundiced patients. (New England J. Med., 16 Oct. '47 - C. L. Holt)

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A Study of Chiniofon Using Radioactive Iodine: Chiniofon, an organic compound containing approximately 27.5 per cent iodine by weight, has been used in the treatment of amebiasis for many years. Despite continued clinical use little is known of its fate in the body.

The purpose of this study was to determine, first, whether absorption occurrs and if so, to what extent and at what rate; second, if absorption occurs, whether it is sufficient to develop blood levels of clinical importance; and third, the pattern of possible urinary excretion.

The availability of radioactive isotopes has made possible the use of the tracer technic in this investigation. Stable iodine ordinarily occurring in the compound was replaced with iodine-131 having an eight-day half-life. Radioactive chiniofon, like the stable form, is easily soluble in water with the addition of a small amount of sodium bicarbonate, forming the sodium salt as it

goes into solution. The compound is bright canary yellow in color and has a bitter taste.

Seven individuals were used as subjects. They varied in age from 30 to 65 years; 2 were female and 5 male; 5 of them were hospitalized for various conditions (mild diabetes, arthritis, Hodgkin's disease) although they were ambulatory and able to cooperate; and 2 were healthy individuals. None had amebiasis.

The drug was given in an aqueous solution of its sodium salt, orally in single doses, ranging in amount from 100 mg. to 300 mg., depending on the intensity of the radioactivity contained.

As a result of these studies it is possible to state that absorption of chiniofon does take place, for it was noted in all of the subjects. The amount absorbed was small, averaging 12.9 per cent of the dose given, and, as might be expected, varied considerably, from 7.46 per cent to 17.5 per cent. This variation is conceivably due in part to individual fluctuations in the rate of passage of tne drug through the intestinal tract. The absorption is prompt as shown by the blood and urinary findings. A peak blood level occurred in two hours following ingestion, and the highest urinary excretion occurred during the initial three-hour period.

Urinary excretion of the drug was found to occur in a pattern similar for all 7 subjects. It is rapid, the bulk being eliminated by 12 hours, and virtually completed in 48 hours. Excretion of the drug is further characterized by its appearance initially in the urine almost entirely intact (averaging for the first 3-hour period 82 per cent intact, as shown by chiniofon-bound iodine, and 18 per cent split-off free iodide and residue, as shown by the difference between total and bound radioactivity) and subsequently reversing this ratio in the second 24-hour period to appear largely split (averaging 27 per cent intact and 73 per cent split). The amount of intact chiniofon excreted through the urine varied from 4.2 per cent to 11.3 per cent of the administered dose, averaging 7.4 per cent.

It appears that fecal excretion plus urinary excretion should equal the total dose of drug given. It is believed that the inability to obtain a total recovery higher than 73 per cent rested probably on technical difficulties involving fecal analyses. (Am. J. Trop. Med., Sept. '47 - E. C. Albright et al.)

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Pigment Changes in Experimental Whole Thickness Skin Grafts: Whether pigment always invades the surrounding tissue after transplantation of skin has not been determined. This report concerns transplantations carried out with large-size grafts.

Fifty black and white guinea pigs weighing from 8 to 13 ounces (227 to 368.5 Gm.) were used. After clipping, the guinea pigs were shaved and washed

and the skin was sterilized with tincture of merthiolate. With a sterile cork borer 17 mm. in diameter, two circular areas of 2.27 sq. cm. were marked out, one in the black and the other in the white skin. The skin so outlined was excised and placed on a piece of gauze moistened with isotonic solution of sodium chloride. After hemostasis had been secured by pressure, the grafts were sutured into place, the black graft in the white area and vice versa. All of the grafts were placed at an angle of variance with the normal direction of the hair fibers. The completed grafts were covered with petrolatum gauze and a moist sea sponge wrapped in gauze, and this dressing was held in place by an adhesive strap. The grafts were undisturbed for 12 days; then the dressings and the sutures were removed, and the progress was noted. In this way, black and white grafts of the same age and subject to the same conditions were observed.

In 11 of the 50 guinea pigs takes of both the black and the white graft were obtained. These 11 animals are the only ones discussed here.

It was noted that during the first 4 weeks following transplantation the grafts shrank to about two thirds or one half of the original size. After the original shrinking stopped, the pigment of the black graft gradually invaded the surrounding white area. This extension of the black pigment continued for as long as 93 days. In direct contrast to this, in all 11 animals the white graft was gradually invaded by the surrounding black pigment until the graft was no longer visible. In 9 of the 11 guinea pigs this invasion of the white graft was completed within from 60 to 80 days. However, in the remaining two guinea pigs, the process took 103 and 129 days, respectively.

The hair growth of the grafts was sparse, since many of the hair cells died during the transplantation. The hair grew out of the grafts in the original direction of growth. It was particularly interesting that in the case of the white grafts white hair continued to emerge from the grafted area for as long as five months, even though the original white graft had become completely pigmented.

The work presented here shows that when a large unpigmented skin graft is transplanted to a colored skin area the surrounding pigment gradually invades the graft. This result is in complete agreement with the earlier work of Loeb and others, who used smaller grafts, and is opposed to that of Seevers and Spencer. As Trotter and Dawson have previously stated, the direction of the hair growth apparently remains constant, being unaffected by the movements and pressure of the surrounding hair growth. (Arch. Path., Aug. '47 - D. E. Barker)

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Benign Pneumoconiosis Due to Metal Fumes and Dusts: The term "pneumoconiosis," first used by Zenker in 1867, requires redefinition because it is so widely misunderstood. Its generic meaning simply is "dust added to the lungs," without any implication of what reaction may or may not have resulted from the dust. Only two dusts, namely, free silica and asbestos, are known to cause significant fibrotic reactions in the lungs. However, because silicosis

and asbestosis are the best known pneumoconioses, the terms "fibrosis" and "pneumoconiosis" unfortunately have become almost synonymous in many medical minds.

The benign pneumoconioses, therefore, are those resulting from the deposits in the lungs of inert dust which does not cause any fibrosis or disability. Siderosis is one of these, as shown in the case reported by Enzer and the author in 1938, in which a tank welder who showed roentgenographic changes simulating silicosis had no fibrous tissue in the lungs when studied at postmortem. The roentgenographic shadows simulating silicosis were shown to be due to aggregations of radiopaque iron particles in the lymphatic channels and nodes surrounding the blood vessels and bronchi.

These findings differ from Zenker's original description of siderosis, in which he implied that the iron dust inhaled into the lungs was the cause of the associated fibrosis. In an analysis of his reported cases it was found that the fibrosis was not due to iron but to associated tuberculosis or silicosis. Also, in analyzing later pathological reports of "siderosis" in iron miners' lungs it was found that the observers had erroneously concluded that the iron, rather than the silica, was the cause of the fibrosis; the disease usually must have been sidero-silicosis. The term "siderosis," therefore, is a perfectly proper one to use for this type of benign pneumoconiosis, provided the implication of fibrosis is excluded.

A number of different occupations appear to have been responsible for the development of siderosis besides the electric welding. In surveys of hematite iron ore miners in England it was first suspected that the hematite itself might be casting some of the shadows seen on the roentgenogram. Fawcitt cites a postmortem study of a hematite miner, made in 1936, in which "no undue fibrosis was found," but aggregations of hematite particles in the pulmonary lymphatics were believed responsible for the "snowflake mottling" seen in the roentgenogram.

More recent postmortem proof that iron oxide particles may form aggregates in the pulmonary lymphatics is offered by McLaughlin, Grout, Barrie and Harding, also of England. In their case a man, aged 54, had been a silver polisher for 40 years during which time "rouge" (Fe_2O_3) was used as the abrasive in his work. The microscopic sections resembled in every detail, including a complete absence of fibrosis, those reported by Enzer and the author in the electric welder in 1938. In the roentgenogram the discrete stippling also resembled that seen in those of the electric welders. These authors attributed largely to siderosis the same roentgenographic appearances which were seen in similar cases among silver polishers.

Hamlin recently described similar roentgenographic changes in foundry grinders and burners, in whom the silica exposure had been insufficient to account for the nodulation. The iron oxide exposure, however, had been sufficiently high to warrant the conclusion that their nodulation was due largely to iron collections in the pulmonary lymphatics. There is no postmortem proof

in these cases. Pendergrass and Leopold have reported 4 cases of suspected siderosis in metal grinders in which by an engineering study they had evidence of insufficient exposure to free silica dust to warrant a diagnosis of silicosis. Another occupation presenting similar exposure factors and roentgenographic appearances is that of boiler scaling, recently reported in England by Dunner, but postmortem proof also is lacking here.

To this growing list of occupations which may be producers of siderosis that of metal cutting with the oxyacetylene flame is added. In roentgenographic surveys of foundry workers dating back to 1933, the author and associates could not explain why these gas cutters working in the foundry cleaning rooms developed nodular silicosis more rapidly than did other employees in the same room. At one time it was suspected that the heat of the flame might be volatilizing some of the adhering molding sand, and therefore it was urged that the castings be more thoroughly cleaned before the cutting operation. It was not realized until after the electric-welder study that the fumes released during the gas cutting of metal contain the same oxides of the metal being cut as those released with electric welding and that these oxides are present as very fine particles (less than 0.5 micron) and in extremely dense concentrations (upwards of 100 million particles per cu. ft.) Although these cutters were known to be inhaling some free silica dust which is present in varying degrees in all foundry cleaning rooms, the silica exposures were too low to account for the nodulation which was appearing in from 6 to 8 years of this work. It is now believed that the nodulation in many of these cases is largely due to siderosis, with little if any associated silicosis. Only a few which appear most representative will be reported here.

Report of Cases. Case I. L. P., aged 35. Oxyacetylene cutter in steel foundry cleaning room since 1933. No previous dust or fume exposure. Chest roentgenogram still clear in 1940 after 7 years of exposure, but gradual development of discrete nodulation or stippling since then. Silica exposure slight, but fume exposure heavy during past 5 years of increased war production. Has no symptoms of any kind and physical findings negative. Will continue at regular work.

Case II. H. O., aged 57. Oxyacetylene cutter in steel foundry cleaning room for past 24 years. No previous dust or fume exposure. Chest roentgenogram in 1933, after twelve years of exposure, shows early discrete nodulation. This has increased only slightly in past twelve years. No symptoms or physical findings in chest. Will continue at regular work.

Case III. J. S., aged 32. Oxyacetylene cutter in steel foundry cleaning room for 10 years (began 1934). Clear lungs to January, 1943, after 9 years of exposure, followed by gradual development of discrete nodulation or stippling. Had attack of "flu" or pneumonia in January, 1944, but precise diagnosis not made and was not roentgenographed then. Next film in March, 1944 showed marked enlargement of root lymph nodes, with persisting dyspnea and cough. Symptoms gradually subsided during next 6 months and root nodes gradually returned to normal size. Has no symptoms now and is working daily. Generalized nodulation or stippling continues.

These 3 cases have the following diagnostic points in common, which are similar in every way to those described for the electric arc welders: (1) discrete and rather sharply defined rounded shadows, of more or less uniform size and equal distribution in both lungs; (2) no tendency to confluence of the shadows; and (3) hilum shadows always smaller than would be expected with silicosis of this degree (except during infections).

In none of these cutters was associated progressive tuberculosis seen, a fact which is further evidence that the cutters had primarily siderosis with little or no silicosis. Non-tuberculous infections, as suggested by the probable pneumonitis and lymphadenitis of Case III, also appeared to clear up completely, indicating no obstruction of the lymphatic circulation. Moreover, there has been a complete absence of symptoms in all of these cases, except during episodes of acute infections. Even in the electric welders, with rather marked roentgenographic changes, there has been no measurable impairment of lung function.

So far there has not been an opportunity for postmortem study of any of these cutters. Some of them may have some silicotic nodulation in addition to the iron pigmentation but, for the reasons given, it is believed that their roentgenographic shadows are due primarily to the aggregations of radiopaque iron particles in the pulmonary lymphatics surrounding blood vessels and bronchi.

It is suggested that all physicians be alert for more postmortem studies of cases in which nodular roentgenographic patterns are seen. Too few have been adequately analyzed and the findings correlated with the roentgenographic appearance. Too often, also, gross black pigmentation at postmortem has been erroneously diagnosed as anthracotic, when ferrocyanide stain of the sections would have made the differential diagnosis by revealing the Prussian blue reaction for iron. Gross round lesions also have been incorrectly diagnosed as silicotic without use of the connective tissue stains to determine if they were true fibrotic nodules. These differential procedures are recommended in all cases in which nodular or stippled roentgenographic shadows were seen.

Finally, more careful analysis of past dust exposures is recommended whenever a roentgenogram showing nodular shadows is presented for an opinion. It is not enough to obtain a history of foundry work, mining, or grinding. The precise environmental conditions must be determined, and this frequently requires sampling of the atmospheric dust and its detailed analysis by an expert industrial hygienist. "Pneumoconiosis" alone no longer can be considered an adequate diagnosis because the benign pneumoconioses must be differentiated from those which may be disabling and progressive. (Am. J. Roentgenol., Sept. '47 - O. A. Sander)

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Early Diagnosis of Uterine Cancer by Vaginal Smear: The vaginal smear test for the early diagnosis of uterine cancer may save many women otherwise condemned to die. A drop of vaginal secretion is obtained by passing a glass

pipet to the posterior fornix of the vagina. Suction is applied by means of an attached rubber bulb. The secretion is blown on a glass slide, stained, and examined for cancer cells.

The authors have recently seen a woman of 45, symptomless except for hot flashes. A supravaginal hysterectomy had been performed 5 years before. The cervix looked and felt normal. A routine vaginal smear was reported positive for cancer, and vaginal cervicectomy was performed. On the excised cervix one laboratory would grant as a pathologic diagnosis only chronic cervicitis. Dr. Arthur Hertig was kind enough to review the slides. He made an unequivocal diagnosis of carcinoma in situ.

Another illustration is this case reported through the courtesy of the Commonwealth Fund Study:

A woman of 40 appeared in the Westfield State Hospital without gynecological complaint. A routine vaginal smear was positive for cancer. A biopsy taken at the time of the first smear showed epidermidization of glands. Biopsies taken 2 and 3 months after the smear were negative. Biopsy 8 months later showed epidermoid carcinoma.

Neither of these two women was suspected of harboring cervical cancer until vaginal smears were examined. In neither was a diagnosis made by examination of the fixed tissue specimen even after the smears were known to be positive. The biopsy remains without question the final diagnostic test. It has not been the authors' policy, except in a few cases, to operate on the basis of a positive smear without biopsy confirmation. The vaginal smear has become, however, a necessary complement to the biopsy. The report of a positive (or doubtful) vaginal smear facilitates and hastens the obtaining of biopsy specimens. It is well known that the biopsy may miss a very early cancer because the involved area may be too small to visualize or may lie in the endocervical canal. Moreover, the biopsy specimen may be so sectioned in the laboratory as to show no epithelium - a technical fault not easy to obviate, especially in small punch specimens. Cancer, under these circumstances, can be neither diagnosed nor excluded. These factors limit the usefulness of the biopsy in the detection of early cancer.

The smear, on the other hand, is not limited to the examination of a small area, but contains cells desquamated from the entire surface including the endocervical canal and the endometrial cavity. Certain limitations, however, also apply to the smear method. First, in some carcinomas, especially those arising in the endometrium, cells which can be recognized as malignant are not found. Cancer cells, moreover, though present in the secretion and even upon the slide, may be missed by the examiner. Again, certain cells, chiefly histiocytes, sometimes normal endometrial and basal cells, may be mistaken for cancer cells. Thus false positive reports may be made. Accurate diagnosis by this method requires training and experience.

In a series of 3720 cases to date, the authors have studied 285 proven carcinomas of the cervix. The vaginal smear was positive in 254., Thus, mistaken negative diagnoses were made in 31 or 10.8 per cent. Of 98 cancers of the endometrium, negative diagnoses were made in 20 - an error of 20.4 per cent. Of the 3327 cases without cancer, a mistaken positive diagnosis was made 55 times - an error of 1.6 per cent.

Judged in relation to other laboratory tests, the vaginal smear is accurate. It should be of greatest value in the screening of large numbers of women for cancer, as the tuberculin test is used to screen for tuberculosis.

Over the past five years, 17 women with proven carcinoma of the cervix were studied in the Vincent Memorial Laboratory. Initial vaginal smears were positive in 15. Perhaps, because in early cases less tumor necrosis has occured, cancer cells are more consistently found in the vaginal smear when small carcinomatous lesions are present than in advanced cases. The method is thus most applicable to just that group for whom a diagnosis offers most hope. The vaginal smear is an important complement to the biopsy in the diagnosis of early cancer of the uterus. (Surg. Clinics of N. America, Oct. '47 - M. Fremont-Smith and R. M. Graham)

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Basic Science Course: The Bureau of Medicine and Surgery announces the availability of a six-months Basic Science Course to be given under the Medical Department adaptation of the Gerow Board Educational System, beginning 5 January 1948. It will be held at the Army Medical Department Research and Graduate School, Washington, D. C.

The purpose of this course is to teach the correlation of the basic medical sciences with the clinical practice of medicine and surgery and their various subspecialties in order to develop an outstanding group of professional specialists. The secondary purpose is to insure adequate instruction in this field so as to enable residents to qualify for examination by the various specialty boards and to pass such examinations with ease.

The first four months will be devoted to general basic sciences which will be given to all specialists.

In the last two months this course is to be made up of special subjects limited to particular specialties having representation among the student body. The individual student will be offered the opportunity to elect a sufficient number of these subjects to occupy the time available and to round out the training in the basic sciences as applied to his particular specialty. For example, the general surgeon will be offered a comprehensive course in gross and microscopic pathology, one in anatomical dissection, one in animal surgery, one in surgical technic, etc.; the internist may be offered courses in anatomy, electrocardiography, experimental medicine, etc.; and the dermatologist and syphilologist will be offered courses in cutaneous pathology, parasitology, mycology, etc.

The quota is one place. Requests are desired from medical officers of the regular Navy and may be made by despatch. No service agreement is required. Requests must reach BuMed prior to 1 December 1947. (Professional Div., BuMed)

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NavMed Dental Forms and Dental Reports: The response of dental activities in the matter of submitting properly accomplished NAVMED dental forms has been highly satisfactory.

In the future, in order to give district and staff dental officers an opportunity to effect the revision and resubmission of incorrect NAVMED dental forms and other dental reports, BuMed will hold the originals of those which have been submitted and which are not properly accomplished until the 20th day of each month, after which they will be forwarded to the cognizant district or staff dental officers with requests for action.

Copies of letters sent by district and staff dental officers for the purpose of effecting the revision and resubmission of improperly accomplished NAVMED

dental forms and dental reports should be forwarded to BuMed so that it may be known in BuMed that action has been taken and that corrected forms and reports may be anticipated. (Dental Div., BuMed)

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Reserve Dental Officers Complete Training in Plastic Ocular Prosthesis: Naval Reserve dental officers from widely separated sections of the United States left their civilian practices for a period of two weeks in order to attend the first course of instruction in Plastic Ocular Prosthesis offered by the Navy especially for Reserve officers. This instruction was presented at the U. S. Naval Dental School between 6 October and 18 October 1947 and was attended by the following Reserve dental officers:

HAWKINS, Jesse F., Lieut., DC, USNR, 317 Citrus Center Bldg., Lakeland, Fla.

HICKS, Thomas J., Jr., Lt. Comdr., DC, USNR, 601 Doctors Bldg., Atlanta 3, Ga.

McAFEE, Chester E., Lieut., DC, USNR, 5337 Greene St., Philadelphia 44, Pa.

POLLACK, Sidney "S", Lt. Comdr., DC, USNR, 5643 N. Fairfield, Chicago 45, Ill.

SCHWARTZ, Irving (n), Lt. (jg), DC, USNR, 1365 Kennedy St., N.W., Washington, D.C.

SUTTON, Glenn A., Lieut., DC, USNR, 1189 Smithfield Ave., Saylesville, R. I.

VOGEL, Stanley W., Lieut., DC, USNR, 1082 Park Ave., New York, New York

WHEELER, George E., Jr., Lt. Comdr., DC, USNR, 886 Main Street, Bridgeport, Conn.

The course was conducted as special training duty. Each Reserve officer was afforded practical experience in constructing a plastic ocular replacement for a patient of the Naval Hospital, Bethesda, Maryland. In addition to clinical experience, lectures and demonstrations in opthalmology, plastic surgery, and maxillofacial prosthesis were included in this course of instruction.

Training in ocular prosthesis was instituted in order to have available dental officers qualified in this specialty in event of a national emergency. (Dental Div., BuMed)

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Reserve Dental Officers' Training Duty at Bethesda: Thirty-six dental officers of the U. S. Naval Reserve from 18 states reported for a two-weeks period of training duty at the U. S. Naval Dental School, National Naval Medical Center, Bethesda, Maryland, on Navy Day, 27 October 1947. An intensive schedule of instruction in naval and dental subjects has been included in the period of training duty. In order to familiarize these Naval Reserve dental officers with typical

organizations of the Navy, field trips have been planned to the U. S. Naval Gun Factory, Washington, D. C., U. S. Naval Academy, Annapolis, Maryland, and U. S. Naval Powder Factory, Indian Head, Maryland. (Dental Div., BuMed)

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Excess Medical and Dental Equipment: Recent inspection of field activities indicates the existence of considerable quantities of medical and dental equipment that is in excess of activity needs.

The Bureau is desirous of having certain of these excess equipment items returned to stock at Naval Medical Supply Depots, provided that such equipment (1) requires minimal or no repairs, and (2) that costs of packing and transportation are not prohibitive.

Excess material must be reported in accordance with procedures contained in Navy Property, Redistribution and Disposal Regulations No. 1, Revised 21 May 1947, Paragraph 801 and Advance Change 5-47, paragraph 302 (c).

When completing column (b) (description) of the reporting form, use particular care to identify the item properly and completely. In many instances the catalog number and title alone are not sufficient. Any variations from the standard catalog description should be noted.

Particular care and judgment must be exercised in the assignment of condition codes to items on the reporting form, W.A.A. Form 1001 (formerly SPB-1), column d. Condition codes are explained in detail in paragraph 801 under Columnar Instructions. When a condition code does not provide an accurate and complete description, it may be omitted and appropriate description of condition entered in column (b).

Adequate attention to the description of an item and the coded or plain language evaluation of its condition will materially assist the Bureau in making a proper decision concerning which items should be returned to stock at Medical Supply Depots. (Materiel Div., BuMed)

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Correct Addresses Desired for BuMed Mail to Inactive Reserves: The records of BuMed indicate that the correct addresses of a number of Reserve officers on inactive duty are not known. The Bureau would therefore like those who do not receive their mail from BuMed at all, or irregularly, or through forwarding, to furnish a record of the address to which they wish material to be sent, to the Publications Distribution Office, Bureau of Medicine and Surgery, Potomac Annex, Navy Department, Washington 25, D. C.

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Official Change No. 2 to the Army-Navy Catalog of Medical Materiel: The following changes, corrections, and deletions shall be made in the Army-Navy Catalog of Medical Materiel upon receipt of this notice:

1. The below listed items are contained in the Addenda to the Catalog as NOT yet available for issue. These items are now available for issue by Naval Medical Supply Depots:

2-003-190 Bandage, Gauze, Compressed, Camouflaged, 4 inches by 6 yards, Brown; with 2 large safety pins. (Navy-for replenishment of Class 9 items).
Note: This item is primarily intended for war-time use in combat areas and for distribution as required for combat missions.

3-103-100 Valve Assembly, with Needle, Puncturing, 15 gage, 1½ inch: With guard and key. Replacement for 3-103-000 and 3-103-560.
Note: For issue to blood bank centers as replacements for 3-103-000 and 3-103-560.

3-452-310 Knife, Skin Grafting, Ferris-Smith: Consists of one handle and one package blades, 3-452-312. Illustrated.
Note: Intended for use by hospitals and hospital ships; maximum stock estimated to be one (1) unit per activity.

3-452-312 Blade, Skin Grafting Knife, Ferris-Smith, 6s: Single edge; 6 inch.
Note: For use with 3-452-310.

3-656-300 Scissors, Tenotomy, Straight, Stevens: Illustrated.
Note: This item is intended for use by hospitals and hospital ships; maximum stock estimated to be three (3) per activity.

3-667-400 Sharpener, Needle, 110 Volt, 60 Cycle, AC: Approximately 150 watts. Complete in case. Illustrated.
Note: This item is intended for use by hospitals and hospital ships.

3-674-700 Snare, Nasal, Krause: With one straight and one angular tube. For snare wire requisition 3-674-780. Illustrated.
Note: This item is intended for use by hospitals and hospital ships; maximum stock estimated to be two (2) per activity.

4-082-300 Bottle, Specimen, Vial, Round, 22 ml: With screw cap. Illustrated.
Note: This item is intended for use in preserving and mailing insects and other specimens by hospitals, hospital ships, large laboratories, epidemiology units, and research units.

4-128-400 Casserole, 340 ml: Porcelain; without lid.
Note: This item is intended for use by large laboratories, hospitals, and hospital ships; maximum stock estimated to be three (3) per activity.

4-314-087	Lubricant, Sealing, 1 oz.: (Celloseal).
Note:	This item is intended for use by all laboratories for the purpose of lubricating and sealing glass stop-cocks and stoppers.
4-365-850	Pipette, Milk: Capacity 17.6 ml. Illustrated.
Note:	This item is intended for use by hospitals, hospital ships, laboratories, epidemiology units, research units, and any other activities having occasion to test milk.
4-425-600	Support, Test Tube, Metal, 20 Tube: Double Shelf, double row. Holds twenty 20 mm. test tubes.
Note:	This item is intended for use by hospitals, hospital ships, large laboratories and research units.
7-123-090	Lamp, Infra-Red, Incandescent Type, 250 watt, 110 volt, AC-DC: 250 watt lamp with built-in reflector and medium screw to fit commercial size lamp sockets. For use with desk, table and similar type lamps.
Note:	This item is intended primarily for use by ships and activities which do not require the larger infra-red units.

2. The below listed items are to be added to the Addenda to the Catalog as items NOT yet available for issue:

9-214-825	First Aid Kit, Life Boat:
9-217-100	First Aid Kit, Life Raft:

3. Make the following corrections as indicated: (Changes appear in quotation marks).

Class	Page	Stock No.	
			Correct description to read "NF" certified
1	2	1-015-525	biological.
1	2	1-050-000	Correct description to read: "NF;" (Absolute alcohol); inflammable.
1	3	1-070-300	Correct description to read: "NF" certified biological.
1	4	1-097-150	Correct description to read: "NF" certified biological.
1	4	1-100-100	Correct description to read: "USP;" the addition etc.
1	4	1-104-000	Correct description to read: "USP".
1	5	1-106-712	Correct description to read: "NF".
1	5	1-106-800	Correct description to read: "NF" certified biological.
1	5	1-106-900	Correct description to read: "NF" certified biological.
1	6	1-125-505	Correct description to read: "NF".
1	6	1-125-515	Correct description to read: "NF." (Navy-For hospitals, etc.)
1	6	1-135-495	Correct Description to read: "NF."

Class	Page	Stock No.	
1	6	1-135-500	Correct description to read: "NF."
1	7	1-160-900	Correct description to read: "NF" certified biological.
1	7	1-162-000	Correct description to read: "NF" reagent grade.
1	9	1-178-010	Correct nomenclature and description to read: Ergot "Fluidextract", 1 oz: "NF".
1	10	1-226-200	Correct description to read: "NF" certified biological.
1	12	1-246-950	Correct description to read: "NF".
1	13	1-290-700	Correct description to read: "NF" certified biological.
1	14	1-301-485	Correct description to read: "USP;" (Nicotinic etc).
1	15	1-323-500	Correct description to read: "NF" certified biological.
1	15	1-325-450	Correct description to read: "USP;" subject to damage, etc.
1	16	1-357-510	Correct description to read: "NF;" powder.
1	18	1-401-000	Correct description to read: "NF."
1	18	1-404-710	Correct description to read: "NF."
1	19	1-429-520	Correct description to read: "NF;" for normal saline, etc.
1	21	1-460-650	Correct description to read: "NF" certified biological; etc.
1	22	1-471-000	Correct description to read: "NF."
1	22	1-474-000	Correct description to read: "NF."
1	23	1-500-625	Correct description to read: "NF."
3	2	3-000-700	Add "LS" in Availability Status column.
3	11	3-175-500	Correct nomenclature and description to read: Catheter, Urethral, Rubber, Hemostatic, "with 30 cc. Bag," 24 Fr: "Foley or balloon type;"hollow tip. Illustrated.
3	11	3-175-510	Correct nomenclature and description to read: Catheter, Urethral, Rubber, Hemostatic, "with 30 cc. Bag," 26 Fr: "Foley or balloon type"; hollow tip.
3	11	3-177-600	Correct nomenclature and description to read: Catheter, Urethral, Rubber, Self-Retaining, "with 5 cc. Bag," 18 Fr: "Foley or balloon type."
3	11	3-177-620	Correct nomenclature and description to read: Catheter, Urethral, Rubber, Self-Retaining, "with 5 cc. Bag," 20 Fr: "Foley or balloon type."
3	11	3-177-640	Correct nomenclature and description to read: Catheter, Urethral, Rubber, Self-Retaining, "with 5 cc. Bag," 22 Fr: "Foley or balloon type." Illustrated.
3	27	3-492-400	Correct nomenclature to read: Needle, Hypodermic, 18 Gage, "1½ inch," 12s: Luer slip.
3	39	3-756-260	Correct Nomenclature to read: Suture, cotton thread, "No. 100","80"yards: White.
7	2	7-002-040	Change description to read: "13 by 25 inches;" etc.

Class	Page	Stock No.	
11	5	11-205-690	Add "LS" in Availability Status column.
11	8	11-302-450	Add "LS" in Availability Status column.
11	8	11-373-150	Add "LS" in Availability Status column.
11	9	11-444-170	Add "LS" in Availability Status column.
11	15	11-877-575	Correct Stock Number to read: "11-877-425".
14	3	14-329-000	Correct description to read: "M860B". etc.
14	20	14-636-000	Correct description to read: "(American Optical Co. No. SC 387A, or equal)." (Navy-Issued, etc.)
14	20	14-637-000	Correct description to read: "(American Optical Co. No. SC 387+A, or equal)." (Navy-Issued, etc.)

4. Delete from Occupational Section, Class 11, the following items:

Page	Stock No.	Nomenclature
3	11-062-250	Pin, Push, Glass Head, 6s:
4	11-131-250	Bench, Work, Bookbinders:
4	11-131-850	Board, Pressing, 10 by 13 inches:
4	11-144-850	Bookbinding Equipment, Hand Operated:
4	11-144-860	Bookbinding Supplies:
4	11-145-180	Pallet, Lettering, Bookbinders:
4	11-145-850	Press, Letter, 10 by 15¼ inches:
4	11-149-850	Tool, Kit, Bookbinding:
4	11-162-350	Cord, Cotton, Macrame, Black, 2 oz.:
4	11-162-365	Cord, Cotton, Macrame, Brown, 2 oz.:
4	11-162-375	Cord, Cotton, Macrame, Dark Green, 2 oz.:
4	11-162-385	Cord, Cotton, Macrame, Navy Blue, 2 oz.:
4	11-162-395	Cord, Cotton, Macrame, Red, 2 oz.:
4	11-162-405	Cord, Cotton, Macrame, Tan, 2 oz.:
4	11-162-415	Cord, Cotton, Macrame, White, 2 oz.:
8	11-302-470	Clay, Pottery, Terra-Cotta, Dry, 5 lb.:
8	11-303-850	Dye, Glaze, Set of 6 colors:
8	11-306-550	Glaze, Transparent, 1 qt.:
8	11-335-550	Fork, Spading:
8	11-336-550	Hoe, Garden, Size 1:
8	11-337-150	Rake, Garden, Level Head, 14 Tooth:
9	11-445-170	Leather, Calfskin, Tan, Tooling, 2 to 2½ oz.:
15	11-877-525	Thread, Cotton, Warp, White, No. 20/2. 20 yards.
15	11-898-220	Yarn, Worsted Wool, Dark Brick Red, No. 4/8s, 4 oz.:
16	11-898-250	Yarn, Worsted Wool, Dark Old Orange, No 4/8s, 4 oz.:
16	11-898-260	Yarn, Worsted Wool, Dark Old Rose, No. 4/8s, 4 oz.:
16	11-898-270	Yarn, Worsted Wool, Dark Taupe, No. 4/8s. 4 oz.:
16	11-898-280	Yarn, Worsted Wool, Dark Wood Brown, No. 4/8s, 4 oz.:
16	11-898-290	Yarn, Worsted Wool, Jade, No. 4/8s, 4 oz.:
16	11-898-330	Yarn, Worsted Wool, Light Old Orange, No. 4/8s, 4 oz.:
16	11-898-390	Yarn, Worsted Wool, Peacock Blue, No. 4/8s, 4 oz.:

5. Delete from Standard Price Supplement the following Stock Numbers:

11-062-250	11-162-375	11-337-150
11-131-250	11-162-385	11-445-170
11-131-850	11-162-395	11-877-525
11-144-850	11-162-405	11-898-220
11-144-860	11-162-415	11-898-250
11-145-180	11-302-470	11-898-260
11-145-850	11-303-850	11-898-270
11-149-850	11-306-550	11-898-280
11-162-350	11-335-550	11-898-290
11-162-365	11-336-550	11-898-330
		11-898-390

6. Delete from Alphabetical Index the following items:

Page	Item Name	Stock No.
8	Bench, Work, Bookbinders	11-131-250
9	Board, Pressing	11-131-850
10	Bookbinding Equipment, Hand Operated	11-144-850
10	Bookbinding Supplies	11-144-860
16	Clay, Pottery	11-302-470
17	Cord, Cotton, Macrame	11-162-350 to 11-162-415
23	Dye, Glaze	11-303-850
29	Fork, Spading	11-335-550
31	Glaze, Transparent	11-306-550
33	Hoe, Garden	11-336-550
49	Pallet, Lettering	11-145-180
51	Pin, Push, Glass Head	11-062-250
53	Press, Letter	11-145-850
69	Tool Kit, Bookbinding	11-149-850
54	Rake, Garden	11-337-150

7. Delete from Numerical Cross Index the following:

Page	Army-Navy Stock No.	Former Army Med 3 Stock No.	Former Catalog of Navy Material, BuMed Stock No.
33	11-062-250	F062250	11-062-250
33	11-131-250	F131250	11-131-250
33	11-131-850	F131850	11-131-850
33	11-144-850	F144850	11-144-850
33	11-145-180	F145180	11-145-180
33	11-145-850	F145850	11-145-850
33	11-162-350	F162350	11-162-350
33	11-162-365	F162370	11-162-365
33	11-162-375	F162380	11-162-375
33	11-162-385	F162360	11-162-385
33	11-162-395	F162390	11-162-395
33	11-162-405	F162400	11-162-405
33	11-162-415	F162410	11-162-415

Page	Army-Navy Stock No.	Former Army Med 3 Stock No.	Former Catalog of Navy Material, BuMed Stock No.
34	11-302-470	F302470	11-302-470
34	11-303-850	F303850	11-303-850
34	11-306-550	F306550	11-306-550
34	11-335-550	F335550	*
34	11-336-550	F336550	*
34	11-337-150	F337150	*
34	11-445-170	F445170	11-445-170
35	11-877-525	F877525	11-877-525
35	11-898-220	F898570	11-898-220
35	11-898-250	F898560	11-898-250
35	11-898-260	F898575	11-898-260
35	11-898-270	F898590	11-898-270
35	11-898-280	F898520	11-898-280
35	11-898-290	F898550	11-898-290
35	11-898-330	F898562	11-898-330
35	11-898-390	F898515	11-898-390

8. Delete from Army Conversion Table the following:

Page	Old Stock No.	New Stock No.	Remarks (See Code)
39	F062250	11-062-250	R5
39	F131250	11-131-250	R5, R8
39	F131850	11-131-850	R5
39	F144850	11-144-850	R4
39	F145180	11-145-180	R4
39	F145850	11-145-850	R5
39	F148910	11-144-860	R4
39	F149850	11-149-850	R4
39	F162350	11-162-350	R5
39	F162360	11-162-385	R5
39	F162370	11-162-365	R5
39	F162380	11-162-375	R5
39	F162390	11-162-395	R5
39	F162400	11-162-405	R5
39	F162410	11-162-415	R5
39	F302470	11-302-470	R5
39	F303850	11-303-850	R5, R8
39	F306550	11-306-550	R5-R8
40	F335550	11-335-550	R5
40	F336550	11-336-550	R4
40	F337150	11-337-150	R5
40	F445170	11-445-170	R5
41	F877525	11-877-525	R5
41	F898515	11-898-390	R4
41	F898520	11-898-280	R4
41	F898550	11-898-290	R4
41	F898560	11-898-250	R4
41	F898562	11-898-330	R4
41	F898570	11-898-220	R4
41	F898575	11-898-260	R4
41	F898590	11-898-270	R4

9. Delete the following from the Navy Conversion Table:

Page	Catalog Navy Material Stock No.	Unit	Army-Navy Stock No.	Unit	Code Symbol	Ratio Equiv.
42	11-062-250	6/pkg.	11-062-250	6/pkg.	R10	
43	11-131-250	Each	11-131-250	Each	R10	
43	11-131-850	Each	11-131-850	Each	R10	
43	11-144-850	Box	11-144-850	Box	R10	
43	11-144-860	Box	11-144-860	Box	R10	
43	11-145-180	Each	11-145-180	Each	R10	
43	11-145-850	Each	11-145-850	Each	R10	
43	11-149-850	Each	11-149-850	Each	R10	
43	11-162-350	2 oz. Bail	11-162-350	2 oz.Bail	R4	#
43	11-162-365	2 oz. Bail	11-162-365	2 oz. Bail	R4	#
43	11-162-375	2 oz. Bail	11-162-375	2 oz.Bail	R4	#
43	11-162-385	2 oz. Bail	11-162-385	2 oz.Bail	R4	#
43	11-162-395	2 oz. Bail	11-162-395	2 oz.Bail	R4	#
43	11-162-405	2 oz. Bail	11-162-405	2 oz.Bail	R4	#
43	11-162-415	2 oz. Bail	11-162-415	2 oz.Bail	R4	#
43	11-302-470	10 lb.Pkg.	11-302-470	5 lb.Pkg.	R3	
43	11-303-850	Set of 6/Pkg	11-303-850	Set	R4	#
43	11-306-550	1 Qt. Can	11-306-550	1 Qt. Can	R10	
44	11-445-170	Skin	11-445-170	Skin	R10	
46	11-877-525	20 Yd.Spl.	11-877-525	20 Yd.Spl.	R10	
46	11-898-220	4 oz.Skein	11-898-220	4 oz.Skein	R10	
46	11-898-250	4 oz.Skein	11-898-250	4 " "	R10	
46	11-898-260	4 " "	11-898-260	4 " "	R10	
46	11-898-270	4 " "	11-898-270	4 " "	R10	
46	11-898-280	4 " "	11-898-280	4 " "	R10	
46	11-898-290	4 " "	11-898-290	4 " "	R10	
46	11-898-330	4 " "	11-898-330	4 " "	R10	
46	11-898-390	4 " "	11-898-390	4 " "	R10	

10. The following changes are to be made effective 15 December 1947:

Change nomenclature and packing to read:

Stock No.	Nomenclature and Description	Unit of Issue	Packaging and Packing
4-060-120	Bottle, Prescription, 1 oz., 72s: With screw cap.	Pkg.	1/6
4-060-150	Bottle, Prescription, 2 oz., 72s: With screw cap.	Pkg.	1/4
4-060-180	Bottle, Prescription, 4 oz., 72s: With screw cap.	Pkg.	1/2
4-060-210	Bottle, Prescription, 8 oz., 36s: With screw cap.	Pkg.	1/2
4-060-240	Bottle, Prescription, 16 oz., 18s: With screw cap.	Pkg.	1/2

Change Unit Price of:

4-060-120 from $.19 to $1.14
4-060-150 from $.22 to $1.32

4-060-180	from $.28 to $1.68
4-060-210	from $.49 to $1.47
4-060-240	from $.61 to $ .90

Change quantities in Navy Component Supplement, Field Kits:

(a) Under Kit No. 9-151-125 change:

4-060-150	Bottle, Prescription, 2 oz., 12s:	Pkg.	1/12
	to read:		
4-060-150	Bottle, Prescription, 2 oz., 72s:	Pkg.	1/72

(b) Under Kit No. 9-248-125 change:

4-060-210	Bottle, Prescription, 8 oz., 12s:	Pkg.	1/12
	to read:		
4-060-210	Bottle, Prescription, 8 oz., 36s:	Pkg.	1/36

(c) Under Kit No. 9-260-125 change:

4-060-120	Bottle, Prescription, 1 oz., 12s:	Pkg.	1
4-060-180	Bottle, Prescription, 4 oz., 12s:	Pkg.	1
4-060-210	Bottle, Prescription, 8 oz., 12s:	Pkg.	1
4-060-240	Bottle, Prescription, 16 oz., 12s:	Pkg.	1
	to read:		
4-060-120	Bottle, Prescription, 1 oz., 72s:	Pkg.	1/6
4-060-180	Bottle, Prescription, 4 oz., 72s:	Pkg.	1/6
4-060-210	Bottle, Prescription, 8 oz., 36s:	Pkg.	1/3
4-060-240	Bottle, Prescription, 16 oz., 18s:	Pkg.	2/3

(d) Under Kit No. 9-483-125 change:

4-060-150	Bottle, Prescription, 2 oz., 12s:	Pkg.	1
4-060-180	Bottle, Prescription, 4 oz., 12s:	Pkg.	1-1/2
4-060-210	Bottle, Prescription, 8 oz., 12s:	Pkg.	1
4-060-240	Bottle, Prescription, 16 oz., 12s:	Pkg.	1
	To Read:		
4-060-150	Bottle, Prescription, 2 oz., 72s:	Pkg.	1/6
4-060-180	Bottle, Prescription, 4 oz., 72s:	Pkg.	1/4
4-060-210	Bottle, Prescription, 8 oz., 36s:	Pkg.	1/3
4-060-240	Bottle, Prescription, 16 oz., 18s:	Pkg.	2/3

(e) Under Kit No. 9-522-675 change:

4-060-210	Bottle, Prescription, 8 oz., 12s:	Pkg.	7/12
4-060-240	Bottle, Prescription, 16 oz., 12s:	Pkg.	9/12
	To Read:		
4-060-210	Bottle, Prescription, 8 oz., 36s:	Pkg.	7/36
4-060-240	Bottle, Prescription, 16 oz., 18s:	Pkg.	1/2

(f) Under Kit No. 9-602-475 change:

4-060-240	Bottle, Prescription, 16 oz., 12s:	Pkg.	1
	To Read:		
4-060-240	Bottle, Prescription, 16 oz., 18s:	Pkg.	2/3

(MatDiv, BuMed)

BuPers Circular Letter 47-179 16 September 1947

To: All Ships and Stations

Subj: Undesirable Discharges Due to Repeated Venereal Infections

Ref: a. Art. D-9112, BuPers Manual

1. Reference (a) contains the instructions and procedures in regard to issuance of undesirable discharges after an individual has demonstrated his unfitness for further retention in the naval service. Among the causes listed is "unclean habits."

2. Men who incur repeated venereal infections are considered to be persons of unclean habits and should be recommended for separation for unfitness following the procedure set forth in reference (a).

3. Separation for unclean habits should apply to all enlisted persons and not to only those whose duties involve the handling or preparation of food.

--BuPers. J. W. Roper

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L20-4/L19(ST) 23 September 1947

To: All Ships and Stations

Subj: Personal Effects of Deceased Personnel, Shipment of

1. Attention is invited to the fact that personal effects of deceased personnel shall be forwarded to the Personal Effects Distribution Center, Naval Supply Depot, Clearfield, Utah, and not to the Naval Supply Depot, Scotia, New York, when there is doubt as to the name and address of the next of kin. The Naval Supply Depot, Scotia, advises that advance bases and vessels continue to forward personal effects of deceased personnel to that activity, which necessitates reshipment from Scotia to the Personal Effects Distribution Center, Naval Supply Depot, Clearfield.

--BuSandA. W. A. Buck

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Circular Letter 47-143 15 October 1947

To: All Ships and Stations

Subj: Hospital Patients, Government Air Transportation of

Refs: (a) BuMed Circ Ltr 46-128 dtd 28 Aug 1946.
(b) MarCorps-BuPers-BuMed Jnt Ltr of 27 Jan 1947, N.D. Bul. of 31 Jan 1947, 47-110.
(c) BuPers-BuSandA Jnt Ltr of 12 Sep 1946, N.D. Bul. of 15 Sep 1946, 46-1887.
(d) BuPers Circ Ltr 209-46, N.D. Bul. of 15 Sep 1946, 46-1887.

1. Reference (a) is cancelled.

2. When transportation for patients is required, it is established policy to use government air transportation for all patients capable of being transported by air both in peacetime and wartime, except when other means of transportation are more expeditious.

3. Hospital flights are regularly scheduled to meet normal patient transport requirements. Special flights will be made from hospitals not on scheduled routes when the number of patients to be transferred or the nature of the case warrants. Requests for special flights shall be addressed to the Bureau of Medicine and Surgery.

4. Naval hospitals and dispensaries desiring air transportation for patients shall obtain authority in accordance with reference (b). After authority has been received they shall request space from the nearest Naval Air Transport Office and provide that office with the following information:

(a) Number of litter patients.
(b) Number of ambulatory patients.
(c) Diagnosis and classification of patients in accordance with Par. 5117.2, Manual of the Medical Department.
(d) Number of female patients, if any.
(e) Date and time transportation desired.
(f) Originating station.
(g) Destination.

5. Flight surgeons and specially trained flight nurses and pharmacist's mates have been assigned to the air transport service to insure adequate medical attendants on all hospital flights both inside and outside the continental limits of the United States. Medical attendants are assigned to each hospital flight as a part of the flight crew. Naval hospitals and naval dispensaries are not required to provide medical attendants except in special cases when sufficient medical attendants cannot be supplied by the air transport service. The originating hospital will honor the request of the air transport service for additional medical attendants, but will not supply attendants unless they have been requested by the air transport office.

6. Upon confirmation of the space by the air transport office, the Medical Officer in Command of the hospital (or the Commanding Officer of the station) shall prepare orders for the patients. In the event additional attendants have been requested by the air transport office, temporary additional duty orders

will be requested from the cognizant issuing command in accordance with reference (c) or (d), as appropriate. These orders shall be so worded that endorsements will not be required en route or at the destination. Orders shall direct travel via government aircraft and authorize per diem. With the exception of psychotic patients, no per diem shall be authorized for the time in transit in the case of inactive, retired, Fleet Reserve and Fleet Marine Corps Reserve personnel. The orders shall state that Class 2 priority via government air is certified for the transportation of patients and their attendants and that a Class 3 priority via government air is certified for the attendants return to their duty station. In the event the attendant has a medical kit his orders shall authorize the excess baggage. Patients on hospital flights will be granted a baggage allowance of one sea bag and one ditty bag, not to exceed sixty-five (65) pounds.

7. Stretcher cases will be delivered on semirigid canvas litters. The originating hospital will supply the litters, sheets, blankets, pillows, pillow cases, and restraining apparatus necessary for the care of its patients on route. An equal number of litters and clean supplies shall be returned by the designation hospital via government air transport.

8. In the case of respiratory paralysis patients the air transport service will deliver a portable respirator to the hospital together with medical attendants instructed in its use. These medical attendants will accompany the patient from the hospital to the patient's destination.

9. The originating hospital will insure that each patient and medical attendant has enough money to provide $3.00 per day for payment for meals and comforts of travel. Facilities for purchase of meals and comforts of travel are provided at stops along the routes, and aboard planes when necessary.

10. The air transport service will advise the destination hospital of the number and classification of patients aboard sufficiently in advance of the plane's arrival to permit ambulance and medical attendants to meet the plane.

11. Reference (b) is not intended to deny government air transportation on regularly scheduled hospital flights of the Naval Air Transport Service to patients transferring from one medical activity to another for his own convenience and not subject to reimbursement. Government air transportation with Class 2 priority may be directed in such cases, but per diem will not be authorized.

MarCorps	BuPers	BuMed
Lemuel C. Shepherd, Jr.	T. L. Sprague	H. L. Pugh

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Circular Letter 47-144 21 October 1947

To: MOinC, U.S. Naval Hospitals

Subj: Form NavMed-103 (rev. 3-47) - Hospital Bed Capacity Quarterly Report - Changes in

This letter from the Chief of BuMed, directs that subject form be modified by (a) adding to table No. 4 a column headed "No. of Qtrs." and (b) changing to "inactive" the word "active" which appears in the instructions on the reverse side of the form in the first sentence under the heading, MAXIMUM EMERGENCY BED CAPACITY - INACTIVE WARD AND PATIENT SPACES.

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Circular Letter 47-145 23 October 1947

To: All Ships and Stations

Subj: Heavy Dental Equipment, Standard Color for

1. The Joint Army Navy Dental Item Review Team has adopted cream color as the standard color for heavy dental equipment. This cream color is: Hue 2 ca, in the Color Harmony Manual of the Container Corporation of America, as established by JAN specifications. When the present Navy stock of green colored equipment is exhausted only cream colored heavy dental equipment will be purchased and issued.

2. The paint on the dental equipment which has been applied by the manufacturer should be retained when possible. Therefore, heavy dental equipment shall not be refinished in cream color only for the purpose of harmonizing it with other equipment. When equipment needs repainting for preservation such refinishing shall be accomplished in the now standard cream color as specified in paragraph 1. As skill and experience are required to properly refinish heavy dental equipment, only those activities that have qualified personnel and the necessary equipment and material available shall undertake to accomplish work of this nature.

3. Standard items of heavy dental equipment now issued by the Naval Medical Supply Depot may be either cream or green in color. Standard models and types of heavy dental equipment presently in use shall be accounted for under Army Navy Catalog of Medical Materiel stock number for standard items regardless of the color they are painted. (For example: A Ritter Senior Dental Operating Unit, Model E, 110 volts, 60 cycle, A.C. is stock number 5-421-475 regardless of the color it is painted).

4. The following are examples of items referred to as heavy equipment:

Stock No.	Items
5-111-005	Cabinet, Dental Instrument.
5-143-000	Chair, Dental Operating
5-174-015	Compressor, Air, with 8 gal. tank, 110V, 60cy, AC
5-175-008	Compressor, Air, with 40 gal. tank, 110V, 60cy, AC
5-385-050	Lamp, Dental, Operating
5-421-475	Operating Unit, Dental, 110V, 60cy, AC
5-513-150	Receptacle, waste.
6-124-920	Radiographic Unit, Dental, Wall mounting, 110V, 60cy, AC

--BuMed. C. A. Swanson

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Circular Letter 47-146 29 October 1947

To: All Ships and Stations

Subj: Yellow Fever Vaccine, Procurement of

Ref: (a) Para. 35B5.2, ManMedDept., Change 2.
(b) Para. 35B20, ManMedDept., Change 2.

1. Yellow fever vaccine may be procured by submitting a separate NavMed-4 requisition to medical supply depots or by letter to the distribution centers listed below, in accordance with reference (a).

Medical Supply Depot, Brooklyn, N. Y.

Medical Supply Depot, Oakland, Calif.

Medical Supply Depot, Navy Supply Center, Pearl Harbor, T. H.

Medical Supply Depot, Naval Supply Center, Guam, M. I.

United States Naval Hospital, Newport, R. I.

United States Naval Hospital, Annapolis, Md.

Dispensary, Puget Sound Naval Shipyard, Bremerton, Wash.

Dispensary, Pearl Harbor Naval Shipyard, Pearl Harbor, T. H.

Dispensary, Naval Air Station, Jacksonville, Fla.

Dispensary, Naval Air Station, Pensacola, Fla.

Dispensary, Naval Air Station, San Juan, P. R.

Dispensary, Naval Training Station, Great Lakes, Ill.

Dispensary, Portsmouth Naval Shipyard, Portsmouth, N. H.

Dispensary, Boston Naval Shipyard, Boston, Mass.

Dispensary, New York Naval Shipyard, Brooklyn, N. Y.

Dispensary, Philadelphia Naval Shipyard, Philadelphia, Pa.

Dispensary, Norfolk Naval Shipyard, Portsmouth, Va.

Dispensary, Charleston Naval Shipyard, Naval Base, S. C.

Dispensary, Naval Training Station, San Diego, Calif.

Dispensary, Submarine Base, Rodman, C. Z.

Dispensary, Naval Station, Guantanamo Bay, Cuba

Dispensary, Washington, D. C.

Post Dispensary. Marine Barracks, Quantico, Va.

2. All ships and stations in the vicinity of the above named activities shall procure their vaccine by having a responsible representative apply for it in person. Advanced base activities shall be supplied from the nearest overseas medical supply depot or storehouse.

3. Reference (b) contains instructions for proper storage and shipment of yellow fever vaccine.

--BuMed. C. A. Swanson

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U. S. Naval Institute of Tropical Medicine Disestablished: By letter dated 3 September 1947, the Acting Secretary of the Navy directed that as of that date the U. S. Naval Institute of Tropical Medicine, Naval Medical Center, Guam, M. I., be disestablished and that the laboratory facilities and functions of this activity, in reduced scope, be incorporated into the U. S. Naval Medical Center, Guam, Marianas Islands.

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